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L28: Entry 15 of 20 File: USPT Sep 28, 1999

US-PAT-NO: 5958769
DOCUMENT-IDENTIFIER: US 5958769 A

TITLE: Compositions and methods for mediating cell cycle progression

DATE-ISSUED: September 28, 1999

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
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US-CL-CURRENT: 435/375; 435/320.1, 435/325, 514/44, 536/23.1, 536/24.5

CLAIMS:

What is claimed is:

1. A method for increasing the proportion of dividing cells in a vertebrate cell population comprising:

exposing said population of cells to an inhibitor of p27 in an amount sufficient to increase the proportion of dividing cells to non-dividing cells relative to said proportion in a population of untreated cells.

2. The method according to claim 1, wherein the cell population is a substantially non-dividing or terminally differentiated primary cell population.

3. The method according to claim 1, wherein the cell population comprises fibroblasts, osteoblasts, myeloblasts, neurons or epithelial cells.

4. The method according to claim 1, wherein the cell population comprises hematopoietic progenitor cells.

5. The method according to claim 1, wherein the exposing step is performed in vitro.

6. The method according to claim 5, further comprising the step of contacting said cell population with a vector which comprises a nucleic acid sequence encoding a desired gene product.

7. The method according to claim 6, wherein the vector which comprises a nucleic acid sequence encoding a desired gene product is a genetically modified virus.

8. The method according to claim 7, wherein the genetically modified viral vector is a retroviral vector.

9. The method according to claim 8, wherein the cell population comprises

hematopoietic progenitor cells.

10. The method according to claim 9, further comprising the step of:

administering the exposed hematopoietic progenitor cells contacted with the retroviral vector to a host for expression of the desired gene.

11. The method according to claim 1, wherein the inhibitor is an oligonucleotide that specifically inhibits p27 expression in said cell population.

12. The method of claim 11, wherein the oligonucleotide is an antisense oligonucleotide.

13. The method according to claim 1, wherein the vertebrate cell is a mammalian cell.

14. The method according to claim 13, wherein the mammalian cell is a human cell.

15. A method for increasing the efficiency of transducing a vertebrate cell population with a viral vector encoding a gene product of interest, comprising:

exposing said population of cells to an inhibitor of p27 in an amount sufficient to increase the proportion of dividing cells to non-dividing cells relative to said proportion in a population of untreated cells, and

contacting said exposed cells to a viral vector encoding the gene product of interest.

16. The method according to claim 15, wherein the vertebrate cell is a mammalian hematopoietic progenitor cell.

17. An inhibitor of p27 which comprises an oligonucleotide that specifically binds to DNA encoding p27 or RNA transcribed therefrom and inhibits expression of p27 protein.

18. The inhibitor of claim 17, wherein the oligonucleotide is an antisense oligonucleotide.

19. An isolated vertebrate cell population which has been treated with an inhibitor of p27 and having an increased proportion of dividing cells to non-dividing cells relative to said proportion in a population of untreated cells.

20. The isolated cell population of claim 19 which comprises hematopoietic progenitor cells.

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L31: Entry 2 of 5 File: USPT Mar 16, 1999

DOCUMENT-IDENTIFIER: US 5882865 A
TITLE: Cancer drug screen based on cell cycle uncoupling

CLAIMS:

1. A method for screening test compounds to identify those which are potential anti-tumor agents, comprising the steps of:

determining DNA content of p21 checkpoint gene-defective human colonic cells incubated in the presence and in the absence of a test compound, wherein a test compound which causes DNA accumulation in the checkpoint gene-defective cells is identified as a potential anti-tumor agent.

7. The method of claim 1 further comprising the steps of:

determining DNA content of p21 checkpoint gene-normal human colonic cells incubated in the presence and in the absence of the potential anti-tumor agent;

identifying a potential anti-tumor agent which preferentially causes DNA accumulation in the checkpoint gene-defective cells as compared to the checkpoint gene-normal cells.

WEST Search History

DATE: Thursday, May 08, 2003

Set Name side by side	Query	Hit Count	Set Name result set
	DB=USPT; PLUR=NO; OP=ADJ		
L72	l71 with l9	2	L72
L71	monitor$3 with (chemotherap$3 or chemotherapeutic$1 or antineoplastic or (anti adj neoplastic) or anticancer or antitumor or antitumor or (anti adj (cancer or tumor or tumour)))	655	L71
L70	monitor$3 with l33	0	L70
L69	L68 or l67	17	L69
L68	L66 and @prad<20000112	1	L68
L67	L66 and @ad<20000112	17	L67
L66	L65 and l56	18	L66
L65	sampl$3[clm]	85618	L65
L64	L63 and l56	2	L64
L63	L62 with l9	43	L63
L62	l51 or l34	3186	L62
L61	L60 and @ad<20000112	8	L61
L60	L59 or l57	8	L60
L59	L56 and l58	1	L59
L58	L51[clm]	323	L58
L57	L56 and l14	7	L57
L56	l3 and L55	181	L56
L55	l9[clm]	597	L55
L54	L52 and @prad<20000112	0	L54
L53	L52 and @ad<20000112	6	L53
L52	L51 near5 apopto$3	11	L52
L51	(tumor or tumour) adj growth adj factor adj (b or beta) or TGFB or TGFbeta or (TGF adj (b or beta))	3186	L51
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L48	L47 and @ad<20000112	5	L48
L47	L46 or l44	59	L47
L46	L45 same apopto$3	40	L46
L45	L39 with l34	157	L45

L44	L40 same l34	44	L44
L43	L41 and @prad<20000112	1	L43
L42	L41 and @ad<20000112	0	L42
L41	L40 with l34	22	L41
L40	L39 with apopto$3	1340	L40
L39	L38 with (treat$4 or therap$5 or inhibit$3 or kill$3 or slow$3 or eliminat$3)	15147	L39
L38	cancer$3 or tumor$3 or tumour$3 or neoplas$3 or malignan$3 or carcinom$5 or leukem$2 or leukaem$2 or lymphom$5 or hodgkin$3	22647	L38
L37	L35 and @prad<20000112	3	L37
L36	L35 and @ad<20000112	0	L36
L35	L34 with l33	37	L35
L34	p21 or p27 or p16	1557	L34
L33	(chemotherap$3 or chemotherapeutic$1 or antineoplastic or (anti adj neoplastic) or anticancer or antitumor or antitumor or (anti adj (cancer or tumor or tumour)))	10914	L33
DB=USPT; PLUR=NO; OP=ADJ			
L32	L29 with l27	7	L32
L31	L30[clm]	5	L31
L30	L29 with l12	76	L30
L29	(chemotherap$3 or chemotherapeutic$1 or antineoplastic or (anti adj neoplastic) or anticancer or antitumor or antitumor or (anti adj (cancer or tumor or tumour)))	37138	L29
L28	L27[clm]	20	L28
L27	l12 with express$3	550	L27
L26	L25 with l12	15	L26
L25	L24 or l23	36534	L25
L24	L5 with therapeutic	34440	L24
L23	L5 with (chemotherap$3 or chemotherapeutic$1 or antineoplastic or (anti adj neoplastic) or therapetuic or anticancer or antitumor or antitumor or (anti adj (cancer or tumor or tumour)))	4359	L23
L22	L20 and l5	52	L22
L21	L20 and l17	0	L21
L20	L12[ti,ab]	99	L20
L19	L16 same l12	0	L19
L18	L16 with l12	0	L18
L17	L16[clm]	1011	L17
L16	L5 with response	8792	L16
L15	L14 and l11	2	L15
L14	L12[clm]	192	L14

L13	L12 and l11	25	L13
L12	p21 or p27 or p16	5487	L12
L11	L8 and l10	172	L11
L10	L9[clm]	597	L10
L9	apopto$3	5278	L9
L8	L6 and l7	5988	L8
L7	L1[clm]	12774	L7
L6	L5[clm]	63597	L6
L5	L4 with (administer$3 or administrat$3 or contact$3)	211932	L5
L4	(treat$4 or therap$5 or inhibit$3 or kill$3 or slow$3 or eliminat$3)	1719051	L4
L3	L2[clm]	6743	L3
L2	L1 with (treat$4 or therap$5 or inhibit$3 or kill$3 or slow$3 or eliminat$3)	46510	L2
L1	cancer$3 or tumor$3 or tumour$3 or neoplas$3 or malignan$3 or carcinom$5 or leukem$2 or leukaem$2 or lymphom$5 or hodgkin$3	81791	L1

END OF SEARCH HISTORY

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L32: Entry 4 of 7 File: USPT Feb 18, 2003

DOCUMENT-IDENTIFIER: US 6521407 B1
TITLE: Methods for determining chemosensitivity of cancer cells based upon expression of negative and positive signal transduction factors

Detailed Description Text (3):
In a similar embodiment, this invention relates to a method for measuring the resistance of cells to the cytotoxic effects of a chemotherapeutic agent, which method comprises testing a sample comprising cells or an extract therefrom for: (a) the level of expression of p21, or for the abundance of p21 protein; and (b) the level of expression of Cyclin D1, or for the abundance of Cyclin D1 protein.

Detailed Description Text (4):
This embodiment also relates to a kit for measuring the resistance of cells to the cytotoxic effects of a chemotherapeutic agent, which kit comprises: (i) a means for testing for the level of expression of p21, or for the abundance of p21 protein; and (ii) a means for testing for the level of expression of Cyclin D1, or for the abundance of Cyclin D1 protein.

Detailed Description Text (5):
Thus, this aspect preferably deals with measuring the levels of Cyclin D1 protein or Cyclin D1 expression, in cells whose p53 mutational status has been determined (e.g. by DNA sequencing) and/or cells displaying substantially undetectable p21 protein levels to determine the resistance of a tumour to, for example, CDDP. High cyclin D1 levels or high cyclin D1 expression, together with p53 mutation or non-elevated (preferably substantially undetectable) p21 protein levels (or substantial non-elevation (preferably substantial lack) of p21 expression), is strongly associated with resistance to chemotherapeutic agents such as CDDP in human cancer cells.

Detailed Description Text (8):
Human cancer cell lines with a combination of p53 mutation or substantially non-elevated (preferably substantially undetectable) p21 protein levels, and high levels of expression of the cyclin D1 protein are resistant to the cytotoxic effects of chemotherapeutic agents such as CDDP. This finding carries important clinical possibilities with regard to providing a potentially new parameter for predictive assays for CDDP responsiveness or a new target for modulating CDDP responsiveness.

Detailed Description Text (42):
In a similar embodiment, the present invention concerns a method for selecting a chemotherapeutic agent for treating cancer, which method comprises: (a) testing a sample comprising cells that substantially do not express p21 and/or in which p21 protein is substantially undetectable, or an extract therefrom for the level of expression of Cyclin D1 or for the abundance of cyclin D1 protein; and (b) if cyclin D1 is overexpressed, and/or cyclin D1 protein is present at elevated levels, selecting for treatment a chemotherapeutic agent comprising a taxane; (c) if cyclin D1 is not overexpressed and/or cyclin D1 protein is substantially not present at elevated levels, selecting for treatment a chemotherapeutic agent comprising an agent other than a taxane.

Detailed Description Text (43):
This embodiment also provides a kit for selecting a chemotherapeutic agent for treatment, which kit comprises: (a) a means for identifying cells in which p21 is substantially not expressed and/or p21 protein is substantially undetectable; and (b) a means for testing for the level of expression of Cyclin D1 or for the abundance of cyclin D1 protein in cells or in a sample therefrom.

Detailed Description Text (44):
Thus, this third aspect preferably deals with a method for selecting a chemotherapeutic agent for treating cancer, which method comprises: (a) testing a sample comprising: (i) p53 mutant cells, or an extract therefrom, (ii) or cells that substantially do not express p21 and/or in which p21 protein is substantially undetectable, or an extract therefrom for the abundance of cyclin D1 protein, and (b) if cyclin D1 is over-expressed, and/or cyclin D1 protein is present at elevated levels, selecting for treatment a chemotherapeutic agent comprising a taxane; (c) if cyclin D1 is not over-expressed and/or cyclin D1 protein is substantially not present at elevated levels, selecting for treatment a chemotherapeutic agent comprising an agent other than a taxane.

Detailed Description Text (46):
Human cancer cell lines with a combination of p53 mutation and high levels of expression of the cyclin D1 protein are resistant to the cytotoxic effects of chemotherapeutic agents such as CDDP, but are sensitive to taxanes. Similarly, human cancer cell lines with a combination of substantially undetectable p21 protein levels and high levels of expression of the cyclin D1 protein are also resistant to the cytotoxic effects of chemotherapeutic agents such as CDDP, but are sensitive to taxanes. These findings carry important clinical possibilities with regard to providing a potentially new parameter for predictive assays for taxane responsiveness or a new target for modulating taxane responsiveness.

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L61: Entry 8 of 8 File: USPT Nov 30, 1999

DOCUMENT-IDENTIFIER: US 5993388 A
TITLE: Nomograms to aid in the treatment of prostatic cancer

Application Filing Date (1):
19980625

CLAIMS:

1. A method for predicting a quantitative probability of recurrence of prostatic cancer following radical prostatectomy in a patient diagnosed as having prostatic cancer comprising the steps of:

correlating a selected set of preoperative factors determined for each of a plurality of persons previously diagnosed with prostatic cancer and having been treated by radical prostatectomy with incidence of recurrence of prostatic cancer for each person of said plurality of persons to generate a functional representation of the correlation, wherein said selected set of preoperative factors comprises pretreatment PSA level, combined Gleason grade and clinical stage, wherein said functional representation of the correlation comprises a pretreatment PSA level scale, a clinical stage scale, a combined Gleason grade scale, a points scale, a total points scale, and a predictor scale, and wherein said pretreatment PSA level scale, said clinical stage scale and said combined Gleason grade scale each have values on said scales which can be correlated with values on the points scale, and wherein said total points scale has values which may be correlated with values on the predictor scale;

determining an identical set of preoperative factors for the patient;

matching the patient's pretreatment PSA level to a corresponding value on the pretreatment PSA level scale, and determining a first point value from the corresponding value on the points scale;

matching the patient's combined Gleason grade to a corresponding value on the combined Gleason grade scale, and determining a second point value from the corresponding value on the points scale;

matching the patient's clinical stage to a corresponding value on the clinical stage scale, and determining a third point value from the corresponding value on the points scale;

adding the first, second and third point values together to get a patient total points value;

matching the patient total points value to a corresponding value on the total points scale; and

correlating the corresponding value on the total points scale with a value on the predictor scale to predict the quantitative probability of recurrence of prostatic cancer in the patient following radical prostatectomy.

11. The method of claim 1 wherein a recurrence of prostatic cancer is characterized as a positive biopsy, bone scan or the application of further treatment for prostate cancer because of the high probability of subsequent recurrence of the cancer.

12. The method of claim 1 wherein the plurality of persons comprises persons with

clinically localized prostate cancer not treated previously by radiotherapy or cryotherapy, and subsequently undergoing radical prostatectomy.

13. The method of claim 1 wherein the selected set of preoperative factors further comprise one or more supplemental factors selected from the group consisting of apoptotic index, maximum cancer length in a core and total length of cancer in the biopsy cores, and said functional representation further comprises one or more supplemental factor scales for each of said one or more supplemental factors, said one or more supplemental factor scales each having values on said scales which can be correlated with the values on the points scale, and wherein said method further comprises the steps of: determining the patient's one or more supplemental factors; matching the patient's one or more supplemental factors to one or more corresponding values on the one or more supplemental factor scales to determine one or more supplemental point values on the points scale; and adding the one or more supplemental point values to the first, second and third point values to determine the patient total points value.

15. The method of claim 14 wherein the selected set of factors further comprises one or more supplemental factors selected from the group consisting of total tumor volume, zone of location of the cancer, p53, Ki-67, p27, level of extraprostatic extension, DNA ploidy status, type of seminal vesicle invasion, clinical stage and lymphovascular invasion and said functional representation further comprises one or more supplemental factor scales for each of said one or more supplemental factors, said one or more supplemental factor scales each having values on said scales which can be correlated with the values on the points scale, and wherein the method further comprises the steps of: determining the patient's one or more supplemental factors; matching the patient's one or more supplemental factors to one or more corresponding values on the one or more supplemental factor scales to determine one or more supplemental point values on the points scale; and adding the one or more supplemental point values to the first, second, third, fourth, fifth and sixth point values to determine the patient total points value.

22. The method of claim 14 wherein a recurrence of prostatic cancer is characterized as a positive biopsy, bone scan or the application of further treatment for prostate cancer because of the high probability of subsequent recurrence of the cancer.

23. An apparatus for predicting a quantitative probability of disease recurrence in a patient with prostatic cancer following a radical prostatectomy, wherein the apparatus comprises: a correlation of preoperative factors determined for each of a plurality of persons previously diagnosed with prostatic cancer and having been treated by radical prostatectomy with incidence of recurrence of prostatic cancer for each person of said plurality of persons wherein said selected set of preoperative factors comprises pretreatment PSA level, combined Gleason grade and clinical stage; and a means for comparing an identical set of preoperative factors determined from the patient diagnosed as having prostatic cancer to the correlation to predict the quantitative probability of recurrence of prostatic cancer in the patient following radical prostatectomy.

27. An apparatus for predicting a quantitative probability of disease recurrence in a patient with prostatic cancer following a radical prostatectomy, wherein the apparatus comprises: a correlation of clinical and pathological factors determined for each of a plurality of persons previously diagnosed with prostatic cancer and having been treated by radical prostatectomy with incidence of recurrence of prostatic cancer for each person of said plurality of persons wherein said selected set of factors comprises preoperative PSA level, specimen Gleason sum, prostatic capsular invasion level, surgical margin status, presence of seminal vesicle invasion, and lymph node status; and a means for comparing an identical set of factors determined from the patient diagnosed as having prostatic cancer to the correlation to predict the quantitative probability of recurrence of prostatic cancer in the patient following radical prostatectomy.

32. The method of claim 30 wherein the adjuvant therapy is selected from the group of radiotherapy, chemotherapy, hormonal therapy, cryotherapy, interstitial radioactive seed implantation, external beam irradiation, hyperthermia, gene therapy, cellular therapy, tumor vaccines, or systemically delivered biologic agents or pharmaceuticals.

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L69: Entry 9 of 17 File: USPT Jun 20, 2000

DOCUMENT-IDENTIFIER: US 6077684 A
TITLE: Automated assay for measuring apoptosis in cell culture

Application Filing Date (1):
19961114

CLAIMS:

1. A method of determining the anti-leukemic activity of a substance, comprising:

a. obtaining a sample of cells from a subject with leukemia;

b. isolating a single cell suspension from the sample;

c. enriching the sample for leukemic cells by removing non-leukemic cells from the sample;

d. placing the enriched leukemic cells in culture;

e. exposing a culture of the enriched cells to the substance;

f. incubating the cultured cells;

g. measuring in a serial manner the optical densities of the culture exposed to the substance;

h. measuring in a serial manner the optical densities of a culture of the enriched cells not exposed to the substance;

i. subtracting at each serial time point the optical densities of the culture of cells not exposed to the substance from the optical densities of the culture of cells exposed to the substance, so as to obtain a net slope of the serially measured optical densities due to the apoptosis-inducing activity of the substance;

j. correlating the slope of a net increase over time in the serially measured optical densities of the cells exposed to the substance with anti-leukemic activity.

2. A method of determining resistance of leukemic cells to an anti-leukemic substance, comprising:

a. obtaining a sample of cells from a subject with leukemia;

b. isolating a single cell suspension from the sample;

c. enriching the sample for leukemic cells by removing non-leukemic cells from the sample;

d. placing the enriched leukemia cells in culture;

e. exposing a culture of enriched cells to the substance;

f. incubating the cultured cells;

g. measuring in a serial manner the optical densities of the culture of enriched cells exposed to the substance;

h. measuring in a serial manner the optical densities of a culture of the enriched cells not exposed to the substance;

i. subtracting at each serial time point the optical densities of the culture of cells not exposed to the substance from the optical densities of the culture of cells exposed to the substance, so as to obtain a net slope of the serially measured optical densities due to the apoptosis-inducing activity of the substance;

j. correlating the absence of a net increase or the presence of a reduced slope of a net increase over time in the optical densities of the culture exposed to the substance with resistance to the substance.

3. A method of determining the relative potential effectiveness of a substance for use in anti-leukemic therapy for a selected subject having leukemia, comprising:

a. obtaining a sample of cells from the subject with leukemia;

b. isolating a single cell suspension from the sample;

c. enriching the sample for leukemic cells by removing non-leukemic cells from the sample;

d. placing the enriched leukemic cells in culture;

e. exposing a culture of the enriched cells to a first selected substance or mixture of the first selected substance and other substances;

f. exposing a culture of the enriched cells to a second selected substance or mixture of the second selected substance and other substances;

g. incubating the cultured cells;

h. measuring in a serial manner the optical densities of the cultures of enriched cells exposed to the first and second substances or mixtures of substances;

i. measuring in a serial manner the optical densities of a culture of the enriched cells not exposed to a substance;

j. subtracting at each serial time point the serially measured optical densities of the culture of cells not exposed to the substance from the optical densities of the culture of cells exposed to the first substance or mixture of substances and the optical densities of the culture of cells exposed to the second substance or mixture of substances, so as to detect differences in the net slopes of the serial optical densities due to differences in the apoptosis-inducing activity of the first and second substances or mixtures of substances;

k. correlating the greater slope of a net increase over time in the serial optical densities of the culture of cells exposed to the first substance compared to the slope of a net increase over time in the serial optical densities of the culture of cells exposed to the second substance with the greater potential effectiveness of the first substance or mixture of the first substance and other substances in anti-leukemic therapy.

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L69: Entry 6 of 17 File: USPT Dec 5, 2000

DOCUMENT-IDENTIFIER: US 6156528 A
TITLE: Methods for using a phosphodiesterase in pharmaceutical screening to identify compounds for treatment of neoplasia

Application Filing Date (1):
19981219

CLAIMS:

1. A method for identifying a compound with potential for treating neoplasia, comprising

determining cyclooxygenase (COX) inhibitory activity of the compound; and

determining cGMP-specific phosphodiesterase ("PDE") inhibition activity of the compound against cGMP-specific PDE enzymatic activity from adenocarcinoma cells;

wherein low COX inhibitory activity and high inhibition of said cGMP-PDE activity identifies that the compound has potential for treating neoplasia.

2. The method of claim 1, further comprising

determining whether the compound inhibits tumor cell growth in a culture;

wherein inhibition of tumor cell growth further identifies that the compound has potential for treating neoplasia.

3. The method of claim 1, further comprising

determining whether the compound induces apoptosis of a tumor cell;

wherein induction of apoptosis further identifies that the compound has potential for treating neoplasia.

4. The method of claim 3, further comprising

determining whether the compound inhibits tumor cell growth in a sample;

wherein inhibition of tumor cell growth further indicates that the compound has potential for treating neoplasia.

5. A method of selecting a compound potentially useful for treating of neoplasia, comprising

determining neoplastic cell growth inhibitory activity of the compound;

determining cGMP-specific PDE inhibition activity of the compound against cGMP-specific PDE enzymatic activity from adenocarcinoma cells; and

selecting a compound that exhibits growth inhibitory activity and said cGMP-specific enzyme inhibitory activity.

6. The method of claim 5, further comprising

determining whether the compound induces apoptosis in a cell; and

selecting a compound that induce apoptosis.

10. A method for identifying a compound potentially useful for administering to patients in need of treatment for neoplasia, comprising the steps of:

determining the COX inhibitory activity of the compound;

determining the cGMP-specific PDE inhibition activity of the compound; and

identifying those compounds for potential use in treating neoplasia in patients in need thereof if the compounds exhibit PDE inhibition activity and have COX inhibitory activity lower that said PDE inhibition activity.

12. The method of claim 11 wherein the growth inhibitory activity is determined by the reduction of the number of cells in a sample.

13. The method of claim 11 wherein the growth inhibitory activity is determined by inducing apoptosis in a sample.

14. A method for identifying a compound with potential for treating neoplasia, comprising:

selecting a compound with cGMP-specific PDE inhibiting activity

evaluating neoplastic cell growth inhibiting activity of the compound; and

identifying the compound that exhibits cGMP-specific PDE inhibiting activity and neoplastic cell growth inhibiting activity wherein said compound has the potential to inhibit neoplasia without substantially inhibiting the growth of normal cells.

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L72: Entry 2 of 2 File: USPT Oct 29, 2002

DOCUMENT-IDENTIFIER: US 6471968 B1
TITLE: Multifunctional nanodevice platform

Detailed Description Text (59):
The present invention also provides the opportunity to monitor therapeutic success following delivery of cisplatin and/or Taxol to a subject. For example, measuring the ability of these drugs to induce apoptosis in vitro is reported to be a marker for in vivo efficacy (Gibb, Gynecologic Oncology 65:13 [1997]). Therefore, in addition to the targeted delivery of either one or both of these drugs to provide effective anti-tumor therapy and reduce toxicity, the effectiveness of the therapeutic can be gauged by techniques of the present invention that monitor the induction of apoptosis. Importantly, both therapeutics are active against a wide-range of tumor types including, but not limited to, breast cancer and colon cancer (Akutsu et al., Eur. J. Cancer 31A:2341 [1995]).